Exhibit "3"

IN THE UNITED STATES BANKRUPTCY COURT
FOR THE DISTRICT OF DELAWARE

In re	*	
W.R. GRACE & CO., *et al.*,	*	Chapter 11 Case No. 01-01139 (JFK) Jointly Administered
Debtor.	*	

GENERAL AFFIDAVIT OF DR. ALAN C. WHITEHOUSE

STATE OF MONTANA)
:ss
County of Lincoln)

DR. ALAN C. WHITEHOUSE, being first duly sworn upon oath, deposes and states as follows:

1. Qualifications.

1. I am Dr. Alan C. Whitehouse. My address is 1507 East Eloika Road, Deer Park, WA 90066.

2. I am licensed in Washington and Montana. I currently practice chest medicine at the Center for Asbestos Related Disease in Libby, Montana where we have about 1,500 active cases of asbestos disease from exposure to Libby tremolite asbestos.

3. My curriculum vitae is attached as Exhibit 1.

4. In addition, I have been an invited speaker on the subject of Libby tremolite asbestos disease at various locations across the country.

1998	Libby, MT	Presentation to local doctors at St. John's Hospital

1/2/00	Bellingham, WA	America College of Occupational and Environmental Medicine
3/00	Cincinnati, OH	Center for Disease Control, meeting on tremolite asbestos disease
5/10/00	Washington D.C.	NIOSH/CDC meeting on tremolite asbestos disease
10/00	Kalispell, MT	Grand rounds at Kalispell Regional Hospital
11/15/01	Butte, MT	ASSE Lecture re Libby asbestos
2001	Washington D.C.	US Senate Hearing Committee Panel, Senator Patty Murray
6/24/02	Missoula, MT	Conference on Asbestos Disease 2002 New Directions and Needs in Asbestos Research
10/4/04	Wenatchee, WA	WA State Public Health Group
11/19/04	Tacoma, WA	ATSDR meeting

5. Since 1980 I have evaluated or treated over 700 patients for asbestos disease from Libby tremolite. Since about 2000, patient data has been tracked on a data base. Since 1980 I have also evaluated or treated over 500 patients for chrysotile asbestos disease. I am in a position to compare asbestos disease from Libby tremolite to asbestos disease from chrysotile asbestos. Chrysotile asbestos is the ordinary form of asbestos used in building materials in the United States, accounting for about 95% of the total asbestos used in the United States. Fraser and Pare's Diagnosis of Diseases of the Chest, 4th Ed. (1999), p.2420.

6. I am Board Certified in internal medicine and pulmonary disease. I treat the entire range of pulmonary disease. In my practice in Spokane in the years 1994-2004, the majority of my time, probably about 90%, was related to general chest disease, including asthma, emphysema, lung cancer and hospital care. About 5-10% of my time was spent on asbestos related issues and other pneumoconioses. Probably about 10% of

my time was related to industrial disease. Currently I spend a small amount of time on legal matters, but for the most part, my time is devoted to patient care.

7. In 30 years of practice I have probably testified at trial 8-12 times, about half for the plaintiff and half for the defendant. I testified in three asbestos trials relating to exposure from the W.R. Grace mine and mill near Libby, and one trial on the same subject in Missoula, Montana. These trials related to asbestos disease from Libby tremolite. In addition, my deposition has been taken on the subject of asbestos disease probably 25-30 times. I have testified in three Libby asbestos cases before the Montana Workers' Compensation Court.

8. I have published a paper on asbestos disease in Libby, titled "Asbestos-Related Pleural Disease Due to Tremolite Associated with Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana," Am J Ind Med 46:219-225 (2004). A copy of the paper is attached as Exhibit 2. 123 patients were followed for an average of 35 months. Lung function was measured in terms of total lung capacity, forced vital capacity and diffusion capacity. The range of loss was between two and four percent per year for each of these functions.

9. Over the last three decades I have practiced occupational medicine. I have performed studies for companies, done screenings for companies and done disability exams for companies. In the 1980s I was involved in multiple screening programs for asbestos disease. I have also done independent medical examinations for the State of Washington, Department of Labor and Industry for decades.

2. The mechanism for asbestos disease.

10. Asbestos is a mineral fiber. There are two kinds, serpentine and amphibole. Serpentine asbestos, or chrysotile asbestos, is the kind used commercially in building products. Serpentine asbestos is more curly, or more club-like, whereas amphibole asbestos is like tiny needles or spears. The Libby asbestos is an amphibole. It is generally referred to as tremolite, and variously referred to as winchite, richterite or tremolite-actinolite, all of which are amphiboles. I will refer to it as tremolite.

11. In relative terms of their length to width (aspect ratio), tremolite fibers are long and sharp, like needles. Some fibers are microscopic, as are the tiny air sacs (alveoli) in the lungs. The fibers when breathed in lodge in the tiny air sacs, and are too small to be expelled. With each breath, they poke and scar the air sacs and the lung tissue structure around the air sacs (the interstitia). Scarring in the interstitia is interstitial disease. When the interstitia are significantly scarred, they can no long expand or contract, and breathing is restricted.

12. The asbestos fibers also migrate through the air sacs to the outside portion of the lung, where they scar and inflame the pleura (the lung lining) and cause pleural disease. See Frazer and Pare, p.2809. This migration seems particularly pronounced with tremolite fibers.

13. The normal pleura is actually thinner than a blown up balloon. It is a very thin membrane, and it can expand like a balloon. Asbestos fiber scarring causes the pleura to look much like the orange portion of an orange rind, and can be just as thick.

When surgeons peel it off the pleura, they call it a rind. When the lung lining becomes more like an orange rind, it can no longer expand freely and breathing is restricted. The correlation between increased pleural thickness and restrictive lung disease is variable in tremolite asbestos disease. Significant restriction can occur with 1mm thickness or more. Asbestos disease is generally a restrictive lung disease.

3. **Diagnosis of asbestos disease.**

14. For the diagnosis of asbestos disease, I use American Thoracic Society (2004), "Diagnosis and Initial Management of Non-Malignant Diseases Related to Asbestos," Am J Respir Crit Care Med, Vol. 170: 691-715 (2004). Asbestos interstitial disease is due to scarring in the lung structure around the alveoli (air sacs) from the poking and inflammation from asbestos fibers. Asbestos pleural disease is due to the scarring and inflamation in the pleura (the lung lining) from asbestos fibers. Asbestos pleural disease and asbestos interstitial disease are essentially the same disease process.

15. The diagnosis of asbestos disease generally requires at a minimum, a history of exposure to asbestos and a 15 year latency period. ATS (2004) states the diagnostic criteria as follows:

> Evidence of structural pathology consistent with asbestos-related disease as documented by imaging or histology.
>
> Evidence of causation by asbestos as documented by the occupational and environmental history, markers of exposure (usually pleural plaques), recovery of asbestos bodies, or other means.
>
> Exclusion of alternative plausible causes for the findings.

16. I have taken hundreds of histories of work exposure at the W.R. Grace

mine and mill near Libby, Montana and am familiar with conditions in the various jobs there. I have also taken hundreds of histories of exposure from family members of workers and Libby community members. Pathways for asbestos disease from Libby tremolite asbestos exposure are discussed at Peipins, et al (2003) "Radiographic Abnormalities and Exposure to Asbestos-Contaminated Vermiculite in the Community of Libby, Montana, USA," Environmental Health Perspectives, 111:14, pp.1753-59.

17. Asbestos disease causes a restrictive defect. The amount of air breathed in is restricted. The physical examination includes determinations of chest restriction, the presence of rales (the crackling sound of scarred air sacs reopening), and an evaluation of shortness of breath. While chest x-rays occasionally show abnormalities not seen on CT scan, chest x-rays generally miss about one-third of parenchymal abnormalities of asbestosis, and miss even higher percentage of pleural abnormalities, as compared to CT scans. See Frazer and Pare, pp. 2440 and 2431, respectively. Subpleural interstitial fibrosis is often not seen on chest x-ray, but is seen on CT scans, and may play a significant role in the severity of the disease process. *See* Schwarz and King, Interstitial Lung Disease, 4th Ed. 2003, p.422.

18. At our clinic, lung function tests are performed in accordance with ATS criteria. We use Knudson norms for vital capacity (spirometry), Intermountain Thoracic Society for lung volumes, and Miller for diffusion capacity.

19. Of all lung function tests, the three most important in asbestos disease are

forced vital capacity (FVC), total lung capacity (TLC) and diffusion capacity (DLCO). Fishman's Pulmonary Diseases and Disorders, 3d Ed. (1998), p.883, states "The characteristic pulmonary function changes of asbestosis are a restrictive impairment with a reduction in lung volumes (especially FVC and total lung capacity) decreased diffusion capacity, and arterial hypoxemia."

20. There are three components to pulmonary function tests. First is spirometry, which measures the amount of volume of the lung and the rapidity of inhalation, which gives an index of air flow and lung volumes. We usually do this before and after brochodilator. If there is improvement with brochodilator, this suggests asthma. There is often an asthmatic effect with asbestos disease from exposure to Libby tremolite asbestos.

Second, we do lung volumes in what is called a body box, or plethysmograph, where we measure very small changes in air flow, pressure and volume, with a shutter and a closed system. Using Boyle's law, one can calculate the volume of the lung.

Third, we measure diffusion capacity, by having the patient breathe a small percentage of carbon monoxide, using very tiny tracer amounts of methane, which is not absorbed, and we measure what comes out of the lungs. We measure the methane, measure the carbon monoxide, and the differential uptake gives us the carbon monoxide diffusion capacity. Diffusion capacity is the efficiency of the lungs in transferring oxygen into the blood stream.

4. Smoking.

21. Smoking causes emphysema and chronic bronchitis.

22. Fishman, p. 684, states:

> The ATS defines **emphysema** as air space enlargement distal to the terminal bronchioles and destruction of the alveolar wall.

23. Fishman, p. 683-684 states:

> The ATS defines **chronic bronchitis** as the persistence of cough and excessive mucus secretions on most days over a three month period for at least two successive years.

24. Fishman, p. 649, states:

> The ATS defines "chronic obstructive pulmonary disease (COPD) as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema."

25. ATS (1995), "Standards for the Diagnosis and Care of Patients with Chronic Obstructive Pulmonary Disease," Am J Respir Crit Care Med, Vol. 152, p.79, states: "Only about 15% of cigarette smokers develop clinically significant COPD."

26. Smoking disease is an obstructive disease. It obstructs what is breathed out. With emphysema, the lung tissue acts like an overexpanded balloon. It does not constrict back to its natural form. Hence exhalation is obstructed.

27. Asbestos disease is generally a restrictive disease. It restricts what is breathed in. The scarring in the lung lining and the lung air sacs and structure restricts the lungs' ability to expand on inhalation.

28. Generally the differences between obstructive disease due to smoking and restrictive disease due to asbestos can be sorted out on pulmonary function tests. This is somewhat complicated by the fact that asbestos disease often causes airway obstruction,

or obstructive disease. A significant obstructive component is often found in the Libby cohort. See Fishman, p.884; Frazer and Pare, p.2445-46.

Fishman, p.568, states:

> The hallmark of the obstructive pattern is a reduction in the FEV1/FVC percentage . . . Typically, all three lung volumes - residual volume, functional residual capacity, and total lung capacity are increased.

Normal for FEV1/FVC is 70 or higher. For hyperinflation in obstructive disease, TLC or RV must be over 120. Fishman, p.569.

5. Tremolite asbestos is highly toxic.

29. Amphibole asbestos in general and tremolite asbestos in particular are far more carcinogenic and fibrogenic (productive of asbestosis) than is chrysotile asbestos. Greenberg (1997), Occupational, Industrial and Environmental Toxicology, p.480 states:

> Several studies have also shown that worker cohorts exposed to higher concentrations of amphibole fibers have higher lung cancer rates than those exposed to similar concentrations of chrysotile asbestos. . . . This pattern of increased toxicity of amphiboles also holds true for all the other asbestos-related lung diseases (asbestosis, pleural disease, and mesothelioma).

30. Fraser and Pare (1999), supra p.1075, states "exposure to amphibole fibers . . . is associated with a significantly greater risk of carcinoma compared to chrysotile exposure."

31. Case (1991), "Health Effects of Tremolite," Annals of NY Academy of Sciences, 491, p.494, states regarding an animal study:

> Significantly, the tremolite fibers were amongst the most carcinogenic tested, with actual incidence of 75% and "percent tumor probability" of 100%.

32. American Thoracic Society (1990), "Health Effects of Tremolite," Am Rev Resp Dis 142:1453, p.1456, states:

Asbestiform varieties of tremolite are highly carcinogenic.

33. Tremolite asbestos is roughly ten times as carcinogenic as chrysotile asbestos. *See* McDonald (1997) "Chrysotile, Tremolite and Carcinogenicity" Annals of Occupational Hygiene, 41:699; *see also*, Antman (1993) "Natural History and Epidemiology of Malignant Mesothelioma," Chest 1993, p.373S, ("Amphiboles are about 10x as carcinogenic as chrysotile.")

34. Tremolite asbestos is roughly five to ten times as fibrogenic as chrysotile asbestos. *See* McDonald (1999) "Chrysotile, Tremolite and Fibrogenicity," Annals of Occupational Hygiene, 43:439. *Compare* Sluis-Cremer (1990) "Evidence for an Amphibole Asbestos Threshold Exposure for Asbestosis," Annals of Occupational Hygiene 34:443 *with* Ontario Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, (1984) Ontario Ministry of the Attorney General, *and* Doll and Peto (1985), "Asbestos: Effects on Health of Exposure to Asbestos," London: Her Majesty's Stationery Office. The following summarizes the above studies' findings re the minimum number of fiber years of exposure (i.e. dose) for asbestosis:

Sluis Cremer (1990)	min 2 fiber years (amphibole)
Doll & Peto (1985)	min 25 fiber years (chrysotile)
Ontario (1984)	min 25 fiber years (chrysotile)
Huang (1990)	min 22 fiber years (chrysotile)

35. The results of the Libby asbestos screening include the following, for pleural abnormalities, two of three B readers concurring:

All participants over 18 (n=6668)	18%
Ever worked for W.R. Grace (n=365)	51%
Lived with W.R. Grace workers (n=1376)	26%
Vermiculite insulation in homes (n=2819)	21%

Peipins, et al (2003) "Environmental Health Perspectives;" 111:14, pp.1753-59. These results clearly indicate that Libby tremolite asbestos is of high toxicity.

36. Amphibole asbestos is more than twice as likely to produce asbestosis and asbestos pleural disease which is more progressive than is chrysotile asbestos. *Compare* Jones (1989) "Progression of Asbestos Effects," British Journal of Industrial Medicine, Gregor (1979) "Radiographic Progression of Asbestosis: Preliminary Report," Annals of the NY Academy of Sciences, and Becklake (1979) "Radiological Changes After Withdrawal From Asbestos Exposure," British Journal of Industrial Medicine, on chrysotile asbestos, *with* Sluis-Cremer (1989) "Progression of Irregular Opacities in Asbestos Miners," British Journal of Industrial Medicine, Cookson (1986) "The Natural History of Asbestosis in Former Crocidolite Workers of the Wittenom Gorge," American Review of Respiratory Disease, Ehrlich (1992) "Long Term Radiological Effects of Third Term Exposure to Amosite Asbestos Among Factory Workers," British Journal of Industrial Medicine, and McDonald (1999) "Chrysotile, Tremolite and Fibrogenicity" Annals Occupational Hygiene, on amphibole asbestos.

37. In most patients with asbestosis (including asbestos pleural disease) from exposure to amphibole asbestos, the asbestosis is progressive. In the words of one author, "it appears that once a dose of asbestos sufficient to initiate the disease has been

retained, it is inexorably progressive." Sluis-Cremer (1989) "Progression of Irregular Opacities in Asbestos Miners," British Journal of Industrial Medicine, 46:846.

38. Cookson (1986) "The Natural History of Asbestosis in Former Crocidolite Workers of Wittenoom George," American Journal of Respiratory Disease 133:994-998, presents Fig. 1, a chart showing that 34 years after first exposure approximately 97% of workers progressed to mild disease, 77% to moderate disease, and 65% to severe disease. Crocidolite, like tremolite asbestos is an amphibole. Based on my experience, I believe the numbers for the Libby workers would be similar, perhaps with a longer lag time.

39. The paper I have published (see ¶ 8 above) demonstrates that Libby tremolite asbestos is highly toxic, and causes highly progressive lung disease.

40. There generally appears to be a distinct pattern for Libby tremolite asbestos disease.

1. The disease appears to be predominately pleural, for the large portion of the time that people have the disease. In my recently published Whitehouse (2004) study, 55% of the patients followed had no interstitial disease and 45% had only minimal interstitial disease. By contrast, chrysotile asbestos disease is mainly interstitial disease. In Libby tremolite asbestos disease interstitial disease becomes radiographically visible rather late in the process, and frequently is only a minor factor.

2. Frequently, we see subpleural interstitial fibrosis on CT scans.

3. The pleural disease is highly progressive leading to restrictive defect and shortness of breath. In June 2002 at the University of Montana Conference on Asbestos Disease, I presented four cases of radiographic progression and death by pure pleural disease. There was no evidence of interstitial disease in these four patients. There are many more patients who demonstrate this pattern. The mechanism for death by pleural disease is loss of pleural space resulting in episodes of hypoxia.

Due to the highly progressive nature of Libby tremolite asbestos disease, once diagnosed with pleural disease, with multiple pleural plaques or diffuse pleural thickening and a loss of lung function, a patient has a high probability of death. In contrast, a patient diagnosed with chrysotile asbestos disease has a much lower likelihood of death.

4. Very often there is an obstructive component associated with Libby tremolite asbestos disease. Obstructive defect has generally been associated with asbestos pleural disease in the literature. See Frazer and Pare, p.2445-46. See also Fishman, p.884. In the Libby cohort it appears to be more pronounced.

5. Pleural pain is often associated with Libby tremolite disease. This is often misunderstood as cardiac chest pain. See Lockey (1984), "Pulmonary Changes after Exposure to Vermiculite Contaminated with Fibrous Tremolite," American Review of Respiratory Disease, (1984) 129:952-958.

6. DLCO (diffusion capacity) is a particularly important indicator of the severity of restrictive disease in the Libby tremolite asbestos disease patients. In the Libby cohort, DLCO shows progressive decline along with FVC and TLC. In some individual cases there is significant asbestos disease on the chest x-ray and only the DLCO is reduced, not the FVC or TLC. Some patients with severe shortness of breath are severe only in the DLCO defect. In my recently published study, Whitehouse (2004), 76% of the 123 patients had progressive loss of lung function. Losses were about the same for FVC, TLC and DLCO at 2-3% per year. DLCO (diffusion capacity) defect is probably associated with subpleural interstitial fibrosis. Whitehouse (2004) explains:

> Pleural changes alone are unlikely to cause a decrease in DLCO. DLCO decreases are likely to be associated with interstitial disease not apparent clinically on either plain chest radiograph or HRCT.

Another amphibole study, Cookson (1983) "Pleural Thickening and Gas Transfer in Asbestosis," Thorax 1983; 38:657-661, notes "The ratio of transfer factor to effective alveolar volume correlated directly with the degree of pleural thickening as alveolar volume fell with increasing severity of pleural disease."

Aspects of the above pattern find support in Lockey (1984), p.956; and in animal studies, Vorwald (1951), "Experimental Studies of Asbestosis," p.32 and Schepers (1955), "An Experimental Study of the Effects of Talc Dust on Animal Tissue," p.322. Other investigators as well have found significant restrictive disease due to pleural thickening. *See* Rom (1992), "Accelerated Loss of Lung Function and Alveolitis in a Longitudinal Study of Non-Smoking Individuals with Occupational Exposure to Asbestos," American Journal of Industrial Medicine, p.843. *See also* Frazer and Pare, p.2446.

6. Latency period.

41. There is a latency period between exposure and the first appearance of asbestos disease on chest x-ray or CT. During the latency period, microscopic asbestos fibers are working at a microscopic level, until they become detectible on chest x-ray or CT. ATS (2000) uses a minimum latency period of 15 years. With tremolite asbestos, the range appears to be about 5-50 years, with an average latency period of about 30-40 years from first exposure to diagnosis.

7. Course of the disease.

42. When asbestos disease due to Libby tremolite exposure is first diagnosable, there usually are no symptoms, only positive findings on chest x-ray or CT. The disease may take decades to progress to a point of severity. Severe disease may include shortness of breath, chest pain, rales, clubbing of the fingernails, hypoxia, cor pulmonale, pleural effusions, and oxygen dependency. *See* ATS (2004). At the end stage, the patient is

bedridden, oxygen dependent, and generally the hypoxia will lead to organ malfunction and death.

8. Workers dead from asbestos disease.

43. In 2000, I performed an evaluation of death certificates and some medical records, and identified 100 workers from the W.R. Grace mine and mill who had died of asbestos disease. Of the 100, 49 died of asbestos lung cancer, 11 died of mesothelioma and 40 died of asbestosis (including asbestos pleural disease). With chrysotile asbestos disease, about 50% of patients with asbestosis develop lung cancer. Frazer and Pare, p.1075. Due to the higher toxicity of tremolite asbestos, the 60% rate of death by asbestos lung cancer and mesothelioma is not surprising.

9. Impairment generally.

44. For Montana cases, I use the <u>AMA Guides to the Evaluation of Permanent Impairment</u> (5th Ed.). I am familiar with it as to lung and heart disease, and recognize it as authoritative. The AMA Guides, p.88, states:

> The purpose of respiratory impairment assessment is (1) to determine if a permanent respiratory impairment exists, (2) quantify its severity, (3) assess its impact on the ability to perform activities of daily living, and, if possible, (4) identify the cause of the abnormality and (5) recommend measures to prevent further impairment and insure proper function. (Numbers added).

Evaluation of pulmonary function tests is the best objective tool in assessing severity of disease. The symptoms suffered in severe disease may include shortness of breath, fatigue, chest pain and cor pulmonale (right sided heart failure).

45. AMA Guides, p.89, presents Table 5-1 "Impairment Classification of Dyspnea (shortness of breath)."

Severity	Definition and Question
Mild	Do you have to walk more slowly on the level than people of your age because of breathlessness?
Moderate	Do you have to stop for breath when walking at your own pace on the level?
Severe	Do you ever have to stop for breath after walking about 100 yards or for a few minutes on the level?
Very severe	Are you too breathless to leave the house, or breathless on dressing or undressing?

46. It is also useful to inquire about shortness of breath upon climbing one flight of stairs. Shortness of breath is a key producer of limitations on physical activities. Often we do an oxygen saturation test by placing an oximeter on the patient's finger and have the patient walk a measured distance or climb a flight of stairs. Normal oxygen saturation at the altitude of 2,000 feet is 93 to 94%, P02 greater than 65, based upon Julius Comroe, Physics of Respiration, p.161. "Desaturation" means an oxygen saturation rate of under 90%. Medicare pays for oxygen at 88% oxygen saturation and below. Desaturation is consistent with severe asbestos disease.

47. AMA Guides, p.89, 5.2 states:

> The significance of respiratory symptoms is better understood when integrated with findings from more objective means, such as physical exam, radiography, lung function and lab studies.

All the above assist in evaluating impairment. Clinical judgment is important in

doing impairment ratings. AMA Guides, p.11, states that "clinical judgment, combining both the "art" and "science" of medicine, constitutes the essence of medical practice."

48. For impairment ratings, the AMA Guides generally rely on criteria presented at page 107, Table 5-12. The Table attempts to apply to many different respiratory disorders, and does not provide a good fit for the restrictive defect found in asbestos disease. As stated above, there are three key pulmonary function test measures for restrictive disease: forced vital capacity, total lung capacity and diffusion capacity. Table 5-12 omits total lung capacity. Total lung capacity can be the most important measure in restrictive disease. The American Thoracic Society, "Lung Function Testing: Selection of Reference Values and Interpretive Strategies," Am Rev Resp Dis 1991; 144:1202-1218, states:

> A restrictive ventilatory defect is characterized physiologically by a reduction in total lung capacity . . . if there is a contradiction between vital capacity and total lung capacity in defining restriction, the classification should be based on total lung capacity.

Interestingly, although the Guides, Table 5-12, omit total lung capacity from the impairment criteria, Table 5-13 includes lung volumes in Respiratory Impairment Evaluation Summary, for restrictive disorders.

49. Lung function test results vary with the individual. Total lung capacity (TLC) may be in the severe range, whereas forced vital capacity (FVC) and diffusion capacity (DLCO) may not, yet the patient may have severe impairment of function. In such cases, the Guides, p.107, call for the use of clinical judgment:

> It is recognized that pulmonary impairment can occur that does not significantly impact pulmonary function and exercise test results but that does impact the ability to perform activities of daily living, such as with bronchiectasis.
>
> In these limited cases, the physician may assign an impairment rating based on the extent and severity of pulmonary dysfunction and the inability to perform activities of daily living (see Table 1-2).

50. We further note that the Guides, Table 5-12, also permit the use of FEV1, as a sole measure of impairment in an asbestos disease evaluation. This is inappropriate, since FEV1 is not a measure of restrictive disease.

51. For forced vital capacity, the Guides do not require use of a brochodiolator. They appear to use the pre-brochodiolator result. I concur with doctors Paul Loehnen and Dana Headapohl of Missoula, Montana, on this point.

52. In addition, Guides, Table 5-12 requires that FVC be in the 40s or DLCO be in the 30s, before the individual is considered impaired greater than 50%. In my experience with patients with asbestos disease from Libby tremolite asbestos exposure, many are dead before they reach this point.

DATED this 15 day of June, 2005.

Dr. Alan C. Whitehouse

SUBSCRIBED AND SWORN to before me this 15 day of June, 2005.

(SEAL)

Candace J. Cummings
Notary Public for the State of Montana
Residing at: Libby, Montana
My Commission Expires: Feb 3, 2006

CURRICULUM VITAE
Alan C. Whitehouse, M.D.

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WIFE:	Sandra
CHILDREN:	Two
EDUCATION:	Cornell University Ithaca, New York BA Chemistry, 1959 University of Cincinnati Cincinnati, Ohio MD 1963
AWARDS	Borden Undergraduate Research Prize-1963 Peter T Kilgore prize -1963- for excellence in medicine Alpha Omega Alpha - honorary medical fraternity 1963
INTERNSHIP & RESIDENCY – INTERNAL MEDICINE:	Duke University Medical Center Durham, North Carolina 1963 – 1965
FELLOWSHIP IN PULMONARY DISEASE & INTERNAL MEDICINE RESIDENCY:	University of Colorado Denver, Colorado 1967 – 1969

EXHIBIT
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MILITARY:	United States Air Force 6571st Aeromedical Research Laboratory Holloman Air Force Base New Mexico 1965 – 1967
BOARDS:	American Board of Internal Medicine, 1970 American Board of Pulmonary Disease 1971
ORGANIZATIONS & SOCIETIES:	Alpha Omega Alpha Member Washington State Thoracic Society Fellow of the American College of Chest Physicians Member of Spokane County Medical Society Member of Washington State Medical Society Member of Spokane Society of Internal Medicine
APPOINTMENTS:	Medical Director Respiratory Therapy Department Sacred Heart Medical Center, Spokane, WA, 1969 – 1989 Past President, Washington Thoracic Society, 1979-1981 Tuberculosis Control Officer Spokane County Health Department – 1978 – 2002 Board of Directors, Spokane Community College Foundation 1984-1988 President, Board of Directors, Spokane Community College Foundation 1988-1990 Washington State Department of Ecology Agricultural Burning Task Force, 1992
PRIVATE PRACTICE:	Continuously 1969-2004 in Spokane (Pulmonary Disease) Associated Internists 1969-1995 Physicians Clinic of Spokane 1995-1998 Drs. Klock & Whitehouse, P.S. 1998 – 2004 CARD / Libby, Mt- 2005- present
LICENSURE:	Ohio, North Carolina, Colorado, Idaho, Washington, Montana

PUBLICATIONS:

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Whitehouse, A. Asbestos-Related Pleural Disease Due to Tremolite Associated with Progressive Loss of Lung Function: Serial Observations in 123 miners, Family Members, and Residents of Libby, Montana. Am J Ind Med 46:219-225, 2004

INVITED PRESENTATIONS ON ASBESTOS

1/2/00	Bellingham, WA	American college of Occupational and Environmental Medicine
3/00	Cincinnati, Ohio	CDC meeting on tremolite asbestos disease
5/10/00	Washington, DC	NIOSH/CDC Meeting on tremolite disease
10/00	Kalispell, MT	Grand Grounds at Kalispell Regional Hospital
2001	Washington, DC	US Senate hearing Senator Patty Murray re asbestos disease
11/15/01	Butte, MT	ASSE Lecture re Libby Asbestos
6/24/02	Missoula, MT	Conference on Asbestos Diseases
10/4/04	Wenatchee, WA	WA State Public Health Group
11/19/04	Tacoma, WA	ATSDR Meeting

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Asbestos-Related Pleural Disease Due to Tremolite Associated With Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana

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Background *The community of Libby, Montana has recently been the focus of national attention secondary to widespread amphibole contamination associated with vermiculite mining and processing.*
Methods *Patients who had occupational and non-occupational exposure to amphibole asbestos in Libby, Montana were evaluated for progressive loss of pulmonary function.*
Results *Of the 123 patients evaluated, 94 demonstrated average age-corrected accelerated loss per year of vital capacity at 3.2%, total lung capacity (TLC) 2.3%, and DLCO 3.3%. All patients all had predominantly pleural changes with minimal to no interstitial disease.*
Conclusions *The study demonstrates a progressive loss of pulmonary function in patients exposed to tremolite asbestos.* Am. J. Ind. Med. 46:219–225, 2004. *© 2004 Wiley-Liss, Inc.*

KEY WORDS: tremolite; asbestos; pulmonary function; Libby; vermiculite; environmental; exposure; mining; dust

INTRODUCTION

In November 1999, it was reported that the community of Libby, Montana was experiencing an epidemic of pulmonary disease associated with occupational and environmental contamination of asbestiform amphibole materials within the community. Investigations revealed that the asbestos contamination was associated with a vermiculite mining and processing operation. Tremolite is an amphibole which has very little commercial value but is a contaminant of the vermiculite ore source in Libby [McDonald et al., 1986a]. This report will reference the high incidence of asbestos related pleural changes and their progression associated with tremolite exposure from the vermiculite mining and processing activity in Libby. The amphibole of the Libby mine has been characterized by mineralogists as a tremolite–actinolite–richterite–winchite transition fiber and will henceforth be referred to as tremolite [US Geological Survey, Bulletin 2193, 2002].

The vermiculite bed seven miles northeast of Libby was discovered in 1916 and mined initially for asbestos by the Zonolite Corporation and then subsequently for vermiculite. It was mined by W.R. Grace & Co. from 1963 to 1990 and was for a long period of time the world's largest producer of vermiculite.

Vermiculite is a hydrated, laminar, aluminum-non-magnesium micacious silicate, which when heated expands to between 10 and 20 times its original proportions and is excellent as an insulator, soil conditioner, and fertilizer additive [Moatamed et al., 1986].

In the process of mining and processing this material, W.R. Grace Company had multiple sites in proximity to Libby including an expanding and shipping facility. The ore body contained 21–26% tremolite and was initially pro-

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EXHIBIT
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cessed on the mountain. The concentrated unexpanded ore, which contained over 2–6% tremolite [Amandus et al., 1987] was then loaded in railcars and shipped throughout the nation to over 200 regional processing or expanding sites. With the application of heat, the ore expands to an accordion like configuration. The expanded vermiculite had up to 1–3% tremolite [Amandus et al., 1987].

Both expanded and unexpanded forms of vermiculite from the mine were made freely available to the community. Many of the homes in the community were insulated with vermiculite. Vermiculite was placed on the ball fields, school track, and children played in piles of vermiculite, which were near the mining and processing facilities. The vermiculite was also used as insulation for plywood dryers in the local lumber mills and could be found in the rail yards where ore cars were loaded for shipping.

Studies of occupational exposure and disease among former vermiculite mine workers found significantly increased rates of asbestosis and lung cancer [Amandus et al., 1987]. A mortality study of the Libby area by the Agency for Toxic Substances and Disease Registry (ATSDR) found that deaths due to asbestosis were among the highest in the country at 40–60 times the expected national rate [DHHS/ATSDR, 2000].

Medical screening in the year 2000 of approximately 6,200 residents of the Libby area who lived there prior to 1990 found over 14% of all participants had radiographic changes consistent with asbestos related abnormalities. These findings represent a significant public hazard in view of the long term health impact known to be associated with amphibole exposure. Additional medical screening in 2001 added more patients, now estimated at over 1,000 plus the 491 patients in this clinical practice who are not part of the 1,000 and who have been followed for up to 14 years. These 491 patients demonstrate isolated pleural plaques to diffuse pleural or interstitial disease including 40 known deaths from asbestos- related diseases. They were examined and followed by a two physician practice specializing in pulmonary disease. The patients were either referred by internists and family practitioners or were self referred. These patients have not been previously reported. Initially, they were mostly employees of W.R. Grace as well as some family members of employees. More recently, non-occupational exposed residents of the community have been identified with asbestos-related health abnormalities. Because of extensive longitudinal medical data in this clinical practice setting, a study was undertaken to determine if there was accelerated loss of pulmonary function in this group of patients.

MATERIALS AND METHODS

Pulmonary function studies including spirometry with bronchodilator, plethysmographic lung volumes, and single breath carbon monoxide diffusion (DLCO) were conducted. The studies prior to 1998 were performed on a Sensormedics model 6200 and subsequently on a Medgraphics model 1085. All studies were done before and after bronchodilator utilizing Albuterol. The same technician was used throughout the entire period. Lung volumes and DLCO were measured after bronchodilator.

Normal values of pulmonary function results used spirometry as described by Knudson et al. [1983], lung volumes established by the Intermountain Thoracic Society [Kanner et al., 1984], and DLCO (non-adjusted values) by Miller et al. [1983]. All studies were reviewed to be certain that height, which was measured to the nearest half inch, and age at test date were correct, and if differences in height were present they were adjusted to match across study dates. American Thoracic Society (ATS) pulmonary function testing guidelines were used throughout [American Thoracic Society, 1995]. In total, 30 patients were removed from the study for the following reasons: chronic obstructive pulmonary disease with elevated residual volumes (14), previous thoracic surgery (1), unacceptable pulmonary function tests because of patient unreliability and inability to meet ATS acceptability criteria (9), and/or the presence of a significant non-asbestos related condition such as sarcoidosis or congestive heart failure (9). Several patients had multiple disqualifying diagnoses. The first and last set of pulmonary function tests were compared for all patients tested (153).

Since the patient values were all age corrected against the normative predicted values, changes in the percentage of predicted over time reflected changes of pulmonary function beyond that accounted for by aging. Differences between the first and last pulmonary function were tabulated and changes per year were calculated. Changes were recorded in percentage change per year because of the wide variation in ages and the usual way of presenting this data in a clinical practice setting.

Repeated measures of analysis of covariance was used to statistically test changes in pulmonary function over time with time modeled linearly. To account for individual differences in the period between assessments, the time between the first and last assessments was entered into the statistical analysis as a covariant.

The initial postero-anterior chest X-ray was graded for extent of pleural changes by the principle investigator and also by a board certified radiologist (Dr. Teel). The extent of pleural changes were graded as follows. The percentage of the lateral chest wall involved with pleural changes was measured and the average of both sides of the chest calculated. All patients were weighed at each visit and body mass index calculated.

RESULTS

Of the 491 subjects, 220 were employees of the vermiculite facilities, 121 were family members, and 150 were

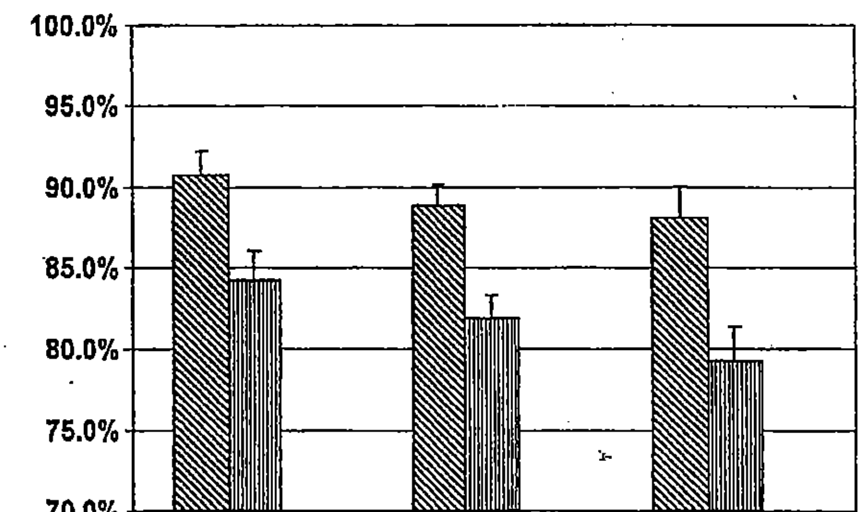
environmental exposures. Two or more sets of pulmonary functions were available on 153 patients. These subjects are representative of the Libby area population and the practice group of 491 patients. All had lived in Libby the majority of their life prior to 1990.

The majority of the 123 patients were ex-smokers with 8 of 123 (7%) being current smokers. Also, 27 (21%) never smoked. In total, 86 (70%) were former employees of W.R. Grace, 27 (22%) were family members of employees, and 10 of 123 (8%) were characterized as Libby environmental exposures only. In total, 99 were males (80%), 24 females (20%), and the average age was 66 years at first pulmonary function study.

Over the course of the study group observation, average BMI increased less than 1 kg/m^2 and there was no statistical correlation between increasing BMI and loss of lung function. Bronchial asthma was also evaluated as a confounding variable. Many subjects used a variety of bronchodilators prescribed by their personal physician although none carried a diagnosis of bronchial asthma and there was no evidence of significant changes in FEV_1 following bronchodilators.

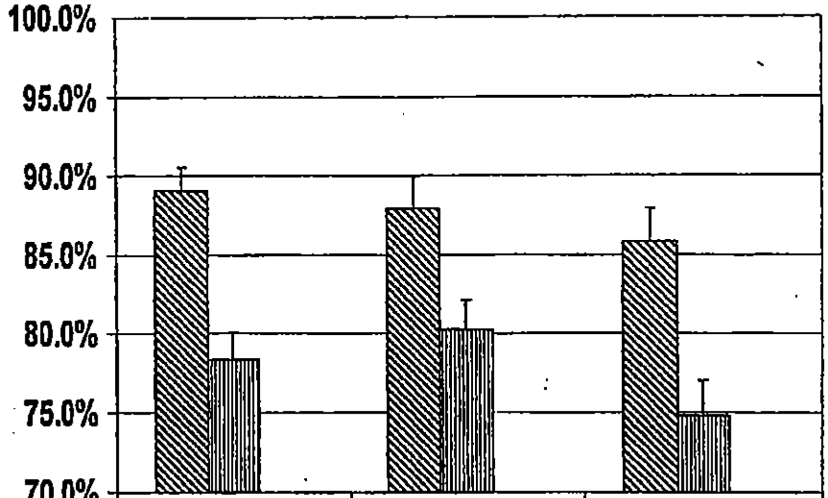
The majority had pleural changes only, consisting of either pleural plaques or diffuse pleural thickening. Because only about half the patients had high resolution computed tomography (HRCT) scans, it was not possible to differentiate this further with any certainty, due to the variations between the plain PA chest film and the HRCT. A total of 67 of 123 (55%) had no evidence on chest X-ray or HRCT of interstitial changes. The remaining patients (56) had minimal radiographic evidence of irregular interstitial changes involving the bases at profusion category 0/1 or 1/0. Of 123 films reviewed, 4 subject films were felt to be normal or equivocal. Of these, all subsequently developed overt pleural changes within a few years and three of four had pleural changes consistent with asbestos exposure on HRCT.

The parameters that were felt to be most valuable for analysis were forced vital capacity (FVC), (taking the best available and valid number from each set), total lung capacity (TLC), and the single breath diffusion capacity (DLCO). In the group of 123 patients (including those with improved FVC), the average yearly loss was 2.2 % for FVC, 2.3% for TLC, and 3.0% for DLCO as calculated over an average of 35 months (Fig. 1). Using FVC as the primary measure of worsening lung function, 94 of the 123 (76%) had an accelerated loss in this parameter. Analyzing the 94 of 123 who had progressive loss of FVC, the loss per year for FVC was 3.2%, TLC 2.3%, DLCO 3.3% (Fig. 2). In total, 79 of 123 patients with greater than 1% loss of FVC per year the average yearly loss was 3.6% for FVC per year, 2.5% for TLC, and 3.5% for DLCO (Fig. 3). The loss rate in this group could not be explained by increases in weight, extent of disease initially or subsequently or other concomitant illness. For the 67 patients with pleural changes alone and with no interstitial changes, the average yearly loss was 2.2% for



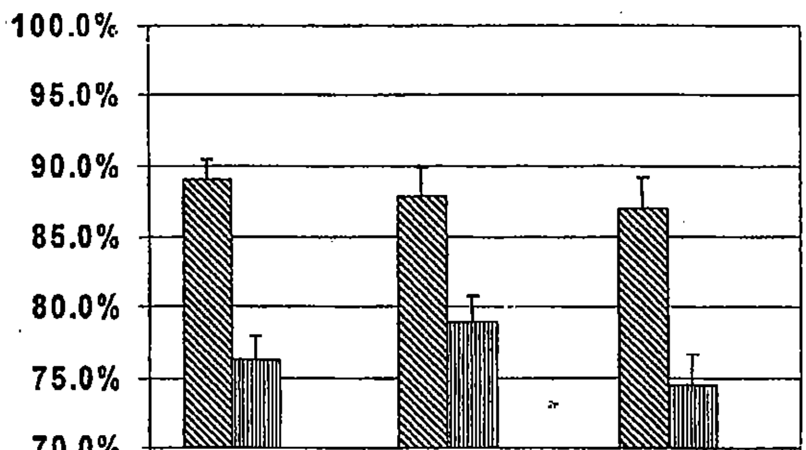
	FVC	TLC	DLCO
BEFORE	90.7%	88.8%	88.0%
AFTER	84.3%	82.0%	79.3%
LOSS/YR	2.2%	2.3%	3.0%

FIGURE 1. Loss of pulmonary function all 123 patients, average 35 months ($P < 0.001$).



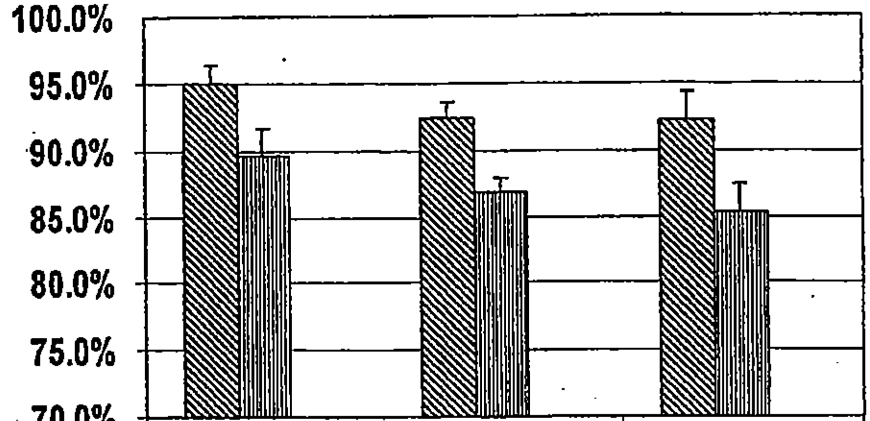
	FVC	TLC	DLCO
BEFORE	89.0%	87.9%	85.8%
AFTER	78.4%	80.3%	74.8%
LOSS/YR	3.2%	2.3%	3.3%

FIGURE 2. Loss of pulmonary function 94/123 patients with worse FVC.



	FVC	TLC	DLCO
BEFORE	89.0%	87.8%	86.9%
AFTER	76.3%	78.9%	74.5%
%LOSS/YR	3.6%	2.5%	3.5%

FIGURE 3. Loss of pulmonary function 79/123 patients with greater than 1% loss rate per year of FVC.



	FVC	TLC	DLCO
BEFORE	95.0%	92.4%	92.2%
AFTER	89.6%	86.8%	85.2%
LOSS/YR	2.2%	2.3%	2.9%

FIGURE 4. Loss of pulmonary function 67/123 patients, pleural disease only.

FVC, 2.3% for TLC, and 2.9% for DLCO (Fig. 4). These results are very similar to those of the entire 123 patients (compare Figs. 1–4).

All values as noted above for decline of pulmonary function were statistically significant at $P \leq .01$. There did not appear to be any difference between the patients with pleural changes who had minor interstitial changes versus no interstitial changes. It is also noted that in the entire group the decline in the diffusion capacity was more rapid than the decline in either the FVC or TLC.

Extent of pleural changes as measured as described on the chest X-ray was evaluated in relation to the loss of lung function. There was no statistical correlation between the extent of pleural changes measured on the chest X-ray and the loss of pulmonary function. The only clearly discernible event leading to accelerated loss of pulmonary function in this entire group was benign asbsestos related effusions (three patients). These were treated vigorously with tube drainage and pleurodysis and the rate of loss equated to the 76% who lost function (2.2–3%).

DISCUSSION

The progressive loss of pulmonary function in 76% of the 123 patients with pleural changes followed in this group of patients with Libby tremolite exposure is excessive compared to other published reports. Progression of asbestos disease in patients with exposure to chrysotile asbestos is well documented. Jones et al. [1989] demonstrated declines in FVC and FEV1 in men who had progressive pleural thickening. Of this group, 31% demonstrated progression of parenchymal small opacities in patients with pleural thickening and smoking was not a significant determinant of pleural progression. The amphibole crocidolite was present in one of the two plants studied and there was a higher rate of progression with crocidolite present. Miller and Miller [1983] demonstrated that patients with longstanding clinically inconsequential plaques remain at risk for diffuse pleural thickening and associated impairment of pulmonary function, which was the case in three patients with pleural effusions. Furthermore, in this group, there was no evidence of progression of small opacities. Decreases in vital capacity have been described by Lilis et al. [1991] and Schwartz et al. [1994]. Ohlson et al. [1985] described 4 year declines in FVC and FEV_1 in a group of asbestos cement workers. The average 4-year decrement of FVC in exposed subjects was 1.9% greater than the reference (control) subjects. Rom [1992] studied 77 asbestos insulators and found that losses of FVC averaged 92 cc per year, FEV_1 66 cc per year, and TLC 14 cc per year. Kouris et al. [1991] found decreased pulmonary function associated with pleural plaques and more significantly with diffuse pleural thickening. Schwartz et al. [1990] demonstrated loss of FEV_1 and FVC associated with both plaques and diffuse pleural thickening and they concluded that "pleural fibrosis" among asbestos exposed patients is an independent predictor of spirometric patterns

consistent with restrictive lung function. Brodkin et al. [1996] further correlates loss of pulmonary function associated with increasing respiratory symptoms. Lockey et al. [1984] described changes in weight as a confounding variable measuring pulmonary function in the workplace. There was no evidence of significant weight changes in this group [McKay et al., 1999].

There are fewer articles on exposure to amphiboles. Shepherd et al. [1997] showed progression of pleural and parenchymal abnormalities associated with amosite. Sluiz-Cremer and Hnizdo, 1989] studied crocidolite workers in South Africa, and was able to demonstrate that once a dose of amphibole asbestos sufficient to initiate disease had been retained it was a naturally progressive process. Cookson et al. [1986] studying crocidolite workers demonstrated that asbestosis was actively progressing even after more than three decades. Erlich et al. [1992] demonstrated in amosite exposed workers that there was progression of pleural abnormalities 20 years after exposure. They found exposure of as little as 1 month was sufficient to produce radiologic signs of parenchymal and pleural fibrosis and progression was detectable greater than 20 years after the end of exposure. McDonald et al. [1986b], studying workers exposed to Libby tremolite from the Grace mine in Libby, Montana, has previously demonstrated extensive pleural plaques and pleural thickening on chest radiographs. Previously, Lockey et al. [1984], was first to describe an association between benign pleural effusions as well as pleural plaques on exposure to Libby tremolite that had been processed at an expansion plant in Ohio to be used as a conditioner for fertilizer.

CONCLUSIONS

This study demonstrates that pleural changes related to exposure to Libby tremolite are associated with progressive loss of pulmonary function in a group of patients exposed to tremolite from approximately 1950 to 1975. Progressive loss of lung function is continuing 40 years after last exposure in 76% of this group who are representative of the population of Libby, Montana. The studies quoted above document both interstitial disease and pleural disease, both radiographically and functionally, but none document the rapid progression of loss of pulmonary function in such a large group of patients with predominantly pleural disease. McDonald et al. [1999] speculated on tremolite's increased fibrogenicity, and it would appear that tremolite–actinolite–richterite–winchite amphibole found in Libby vermiculite has a propensity for causing pleural changes that result in a progressive restrictive pattern on pulmonary function testing. Pleural changes alone are unlikely to cause a decrease in DLCO. DLCO decreases are likely to be associated with interstitial disease not apparent clinically on either plain chest radiograph or HRCT.

Exposure histories for this group are complex, because for the most part there was continuous exposure throughout this entire period that they lived in Libby, whether they were mine workers, family members of workers, or community members living near the vermiculite processing facilities.

This study demonstrates that the number of patients progressing is much higher than has previously been reported in studies with either chrysotile or amphibole asbestos exposure. Lincoln County, Montana, (where Libby is the county seat) has the highest mortality rate from asbestosis in the nation [DHHS/ATSDR CERCLIS No MT00090883840, 2000].

It is apparent from these data that the majority of the 1,500 persons who have radiologic changes of asbestos exposure are at increased risk for progressive loss of lung function from pleural changes alone or from potential future development of interstitial fibrosis. Assuming a latency period of between 20 and 30 years to significant disease, it is not unreasonable to expect that the people of Libby, Montana will have to be monitored over the next 30–40 years, because of the risk for loss of pulmonary function and other known diseases historically associated with asbestos exposure.

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